TAB A

Recommended Dietary Allowances

10th Edition

The most authoritative source of information on nutrient allowances for healthy people.

NATIONAL RESEARCH COUNCIL

Recommended Dietary Allowances

10th Edition

Subcommittee on the Tenth Edition of the RDAs
Food and Nutrition Board
Commission on Life Sciences
National Research Council

NATIONAL ACADEMY PRESS
Washington, D.C. 1989

FOOD AND NUTRITION BOARD SUBCOMMITTEE ON THE TENTH EDITION OF THE RDAS

RICHARD J. HAVEL *(Chairman)*, Cardiovascular Research Institute, University of California, San Francisco, California
DORIS H. CALLOWAY, Department of Nutritional Sciences, University of California, Berkeley, California
JOAN D. GUSSOW, Department of Nutrition Education, Teachers College, Columbia University, New York, New York
WALTER MERTZ, Human Nutrition Research Center, Agricultural Research Service, U.S. Department of Agriculture, Beltsville, Maryland
MALDEN C. NESHEIM, Provost, Cornell University, Ithaca, New York

Food and Nutrition Board Staff
SUSHMA PALMER, *Director*
PAUL R. THOMAS, *Program Officer*
FRANCES M. PETER, *Editor*
ALDON GRIFFIS, *Research Assistant*
MARIAN F. MILLSTONE, *Research Assistant*

The Food and Nutrition Board wishes to acknowledge the contributions of the Committee on Dietary Allowances that worked on the tenth edition from 1980 to 1985. The committee was chaired by the late Henry Kamin of Duke University Medical Center. Other members of the committee were Philip Farrell (University of Wisconsin, Madison), Helen A. Guthrie (Pennsylvania State University), Victor Herbert (Veterans Administration Medical Center, Bronx, New York), Robert Hodges (University of California, Irvine Medical Center), Max K. Horwitt (St. Louis University School of Medicine), Orville Levander (U.S. Department of Agriculture), Hellen Linkswiler (deceased; University of Wisconsin, Madison), James A. Olson (Iowa State University), and Peter L. Pellett (University of Massachusetts, Amherst). The Food and Nutrition Board staff included Myrtle L. Brown (executive secretary until August 1983), Sushma Palmer (director from July 1983), Linda D. Myers (project director), Frances M. Peter (editor), Marianne E. La Veille (research associate), and secretaries Susan Barron, Shirley E. Cole, Avis I. Harris, and Janie B. Marshall.

Venkatachalam, P.S., B. Belavady, and C. Gopalan. 1962. Studies on vitamin A nutritional status of mothers and infants in poor communities of India. J. Pediatr. 61:262–268.

Villard, L., and C.J. Bates. 1987a. Dietary intakes of vitamin A precursors by rural Gambian pregnant and lactating women. Hum. Nutr. Appl. Nutr. 41A:135–145.

Villard, L., and C.J. Bates. 1987b. Effects of vitamin A supplementation on plasma and breast milk vitamin A levels in poorly nourished Gambian women. Hum. Nutr. Clin. Nutr. 41C:47–58.

Wagner, K.H. 1940. Die experimentelle Avitaminose A beim Menschen. Seyler's Z. Physiol. Chem. 264:153.

Wallingford, J.C., and B.A. Underwood. 1986. Vitamin A deficiency in pregnancy, lactation, and the nursing child. Pp. 101–152 in J.C. Bauernfeind, ed. Vitamin A Deficiency and its Control. Academic Press, New York.

WHO (World Health Organization). 1982. Control of Vitamin A Deficiency of Xerophthalmia. A Report of a Joint WHO/UNICEF/USAID/Helen Keller International/IVACG Meeting. Technical Report Series No. 672. World Health Organization, Geneva. 70 pp.

Wolf, G., and B.C. Johnson. 1960. Metabolic transformations of vitamin A. Vitam. Horm. 18:403–415.

Ziegler, R.G., T.J. Mason, A. Stemhagen, R. Hoover, J.B. Schoenberg, G. Gridley, P.W. Virgo, and J.F. Fraumeni, Jr. 1986. Carotenoid intake, vegetables, and the risk of lung cancer among white men in New Jersey. Am. J. Epidemiol. 123:1080–1093.

VITAMIN D

Vitamin D (calciferol) is essential for the proper formation of the skeleton and for mineral homeostasis. Exposure of the skin to ultraviolet light catalyzes the synthesis of vitamin D_3 (cholecalciferol) from 7-dehydrocholesterol. The other major form of the vitamin, D_2 (ergocalciferol), is the product of the ultraviolet light-induced conversion of ergosterol in plants. The effectiveness of exposure to sunlight or ultraviolet light in curing or preventing rickets was shown early in the twentieth century (Chick et al., 1923).

General Signs of Deficiency

Vitamin D deficiency is characterized by inadequate mineralization of the bone. In children, severe deficiency results in deformation of the skeleton (rickets). In the adult, vitamin D deficiency leads to undermineralization of the bone matrix osteoid; the resulting hypocalcemia is accompanied by secondary hyperthyroidism that can lead to excessive bone loss and, in the extreme, bone fractures (osteomalacia) (Nordin, 1973). The prolonged periods required to produce vitamin D deficiency in animals and humans is attributed to the slow release of vitamin D-related steroids from fat depots and skin.

Because milk and other foods are fortified with vitamin D, rickets is very rare in many countries. However, vitamin D deficiency occurs in some infants who are breastfed without supplemental vitamin D or exposure to sunlight (Edidin et al., 1980; Hayward et al., 1987), in the elderly (Egmose et al., 1987; Omdahl et al., 1982; Reid et al., 1986), and in people with vitamin D malabsorption (Rosen and Chesney, 1983). Abnormalities in calcium homeostasis and bone metabolism can also occur when the conversion of vitamin D to biologically active forms is compromised due to disease states. For example, rickets and osteomalacia are often found in patients with kidney failure (Haussler and McCain, 1977).

The Dietary Essentiality of Vitamin D

The vitamin D requirement of humans can be met if their skin is exposed to a sufficient amount of sunlight or artificial ultraviolet radiation. The amount of vitamin D synthesized by this means is dependent on the area of skin exposed, the time of exposure, and the wavelength of the ultraviolet light impinging on the skin. Practical considerations are the latitude of the person's residence and the season of the year (Lawson, 1980; Webb et al., 1988). Exposure to sunlight can be further limited by customs of dress and by the institutionalization and extensive indoor residency of the ill and aged. The character of the skin also influences the efficiency of vitamin D_3 synthesis. Compared to lighter skin, skin with high melanin content requires a much longer exposure to ultraviolet light to achieve the same degree of synthesis (Clemens et al., 1982). The capacity of skin to synthesize vitamin D_3 in the elderly is approximately half that of younger people (Webb et al., 1988). Given the many factors that can affect the magnitude of ultraviolet light-dependent synthesis of vitamin D_3, vitamin D should be considered an essential dietary nutrient.

Biochemistry and Metabolism

The biochemistry and metabolism of vitamin D have been extensively reviewed (DeLuca, 1988; Fraser, 1988). Among the metabolites of vitamin D are 25-hydroxyvitamin D [25(OH)D, or calcidiol], which is formed in the liver and further hydroxylated in the kidney to yield 1,25-dihydroxyvitamin D [$1,25(OH)_2D$, or calcitriol], and 24, 25-dihydroxyvitamin D [$24,25(OH)_2D$]. In addition to ensuring adequate absorption of calcium, $1,25(OH)_2D$ contributes to plasma calcium regulation by increasing bone resorption synergistically with para-

thyroid hormone and stimulating the reabsorption of calcium by the kidney.

Dietary vitamin D is readily absorbed from the small intestine and transported in chylomicrons to the liver, where conversion to 25(OH)D takes place (DeLuca, 1979). Vitamin D from the liver and vitamin D synthesized in the skin are transported in the blood largely bound to a vitamin D-binding protein and albumin, as are 25(OH)D and $1,25(OH)_2D$. The liver is the major site of vitamin D deactivation. Some of the metabolites of the vitamin excreted in bile are reabsorbed, but this process contributes little to the maintenance of vitamin D status.

Vitamin D status is reflected primarily by the concentrations of 25(OH)D and $1,25(OH)_2D$ in the blood. In surveys of large groups of healthy people, the mean value of 25(OH)D ranges from approximately 25 to 30 ng/ml (Rosen and Chesney, 1983). The concentrations of $1,25(OH)_2D$ range from 18 to 60 pg/ml of plasma in normal children and between 15 to 45 pg/ml in healthy adults. Despite the wide range of normal values, there is no seasonal variation in plasma $1,25(OH)_2D$ (Chesney et al., 1981); this implies tight regulation.

One international unit (IU) of vitamin D is defined as the activity of 0.025 μg of cholecalciferol in bioassays with rats and chicks. Thus, the biological activity of cholecalciferol is 40 IU/μg. The activity of 25(OH)D and $1,25(OH)_2D$ are approximately 1.5 and 5 times, respectively, greater than that of vitamin D.

Dietary Sources and Usual Intakes

In the United States, foods fortified with vitamin D are a major dietary source of the vitamin.[a] Processed cow's milk, which contains 10 μg of cholecalciferol (400 IU) per quart, contributes most of the vitamin ingested by children. Infant formulas are fortified with the same amount as milk. Human milk contains 0.63 to 1.25 μg of cholecalciferol per liter (Reeve et al., 1982; Tsang, 1983). The usual solid food sources are eggs, butter, and fortified margarine. The vitamin is stable in foods. Storage, processing, and cooking do not appear to affect its activity.

In the United States, the usual dietary intake has been estimated primarily for infants and children. Calculations based on reference

[a]Vitamin D occurs as cholecalciferol or ergocalciferol in foods and fortified food products. Since the chemical forms are generally not separately identified, the vitamin D content of foods and dietary intakes are given in micrograms of cholecalciferol for simplicity.

infants and the data of Fomon (1974) indicate that daily intakes of vitamin D from formula are 6.75 μg of cholecalciferol for the infant from birth to 3 months of age and 8.5 μg of cholecalciferol at 4 to 6 months. In contrast, the average breastfed reference newborn receives only 0.38 to 0.75 μg of cholecalciferol per day from 750 ml of human milk (AAP, 1985; Reeve et al., 1982). Children drinking three 8-oz glasses of milk daily consume about 7.5 μg of cholecalciferol plus a small amount in other foods. Data from the USDA show that the average adult male ingested 2.1 μg of cholecalciferol from milk (USDA, 1986), whereas females consumed 1.5 μg (USDA, 1987). Omdahl et al. (1982) reported that a population of 60- to 93-year-old subjects had a median dietary intake of 1.35 μg of cholecalciferol (females) and 1.95 μg of cholecalciferol/day (males); 15% of the total study population, especially women, had plasma 25(OH)D levels suggestive of deficiency.

Recommended Allowances

Establishing an RDA for vitamin D is difficult because exposure to sunlight results in synthesis of vitamin D by the skin. People regularly exposed to sunlight, under appropriate conditions, have no dietary requirement for vitamin D. However, since a substantial proportion of the U.S. population is exposed to very little sunlight, especially during certain seasons (Stryd et al., 1979), a dietary supply is needed.

Adults Data to assess vitamin D requirements of adults are limited. Dent and Smith (1969) summarized studies of seven adult females living in the United Kingdom and suffering from nutritional osteomalacia due to vitamin D deficiency. They were either strict vegetarians or had unusual diets that rigidly excluded most fats. In all the patients, vitamin D intake was estimated to be below 1.75 μg (70 IU) per day and small additional amounts of vitamin D resulted in improved calcium utilization. On the basis of these studies and other observations on similar patients, Dent and Smith suggested that the adult vitamin D requirement was about 2.5 μg (100 IU) per day.

The relative paucity of recent controlled studies in humans and the lack of data on the variability of vitamin D requirements have led this subcommittee to keep the RDA for vitamin D for adults beyond 24 years of age at 5 μg (200 IU)—the same level recommended in 1980. It seems likely that this is a generous allowance. Data from USDA's 1977–1978 Nationwide Food Consumption Survey indicate that 1.25 to 1.75 μg/day (50 to 70 IU) is the usual dietary intake in the United States (USDA, 1983). Presumably, vitamin D

stores are enriched in most people by regular exposure to sunlight, at least during certain times of the year. Clinical nutritional osteomalacia appears to be rare in the United States.

Pregnancy and Lactation It has not been determined whether or not there is an increased need for vitamin D during pregnancy, but since calcium is deposited in the growing fetus, a daily increment of 5 μg (200 IU) is recommended for women beyond 24 years of age. Although only small amounts of vitamin D are secreted in human milk, an increment of 5 μg (200 IU) per day is recommended for lactating women beyond age 24 because of the importance of maintaining calcium balance. The vitamin D RDA for both pregnant and lactating women of all ages is 10 μg/day (400 IU).

Infants and Children Several reports have questioned whether human milk contains sufficient vitamin D to prevent rickets in the absence of exposure to sunlight (Finberg, 1981; Greer and Tsang, 1983; Tsang, 1983). In full-term infants fed human milk, bone mineral content, total and ionized calcium in serum, and serum phosphorus and alkaline phosphatase values were similar to those in a comparison group fed infant formula containing 10 μg (400 IU) of vitamin D per quart, but serum 25(OH)D concentrations were lower in the babies fed human milk (Roberts et al., 1981). In a randomized, double-blind study, bone mineral content was less in babies fed human milk without supplemental vitamin D than in those who received 10 μg/day (400 IU) (Greer et al., 1982). In a study of premature infants, 2.5 μg (100 IU) of vitamin D daily was associated with rickets and abnormalities in alkaline phosphatase activity in some infants (Glaser et al., 1949); however, these abnormalities may have been due to dietary mineral deficiency (Steichen et al., 1981). To provide a margin of safety, the RDA is set at 7.5 μg (300 IU) for infants from birth to 6 months of age. Breastfed infants who are not exposed to sunlight should receive a daily supplement of 5 to 7.5 μg (200 to 300 IU).

The allowance for children older than 6 months of age has been set at 10 μg (400 IU) because of their increased body mass. Because peak bone mass is not achieved before the third decade, this allowance is recommended through age 24 years. This amount should be readily achievable at current levels of vitamin D fortification of foods.

Excessive Intakes and Toxicity

Vitamin D is potentially toxic, especially for young children. The effects of excessive vitamin D intake include hypercalcemia and hy-

percalciuria (Haussler and McCain, 1977), leading to deposition of calcium in soft tissues and irreversible renal and cardiovascular damage. Although the toxic level has not been established for all ages, consumption of as little as 45 μg (1,800 IU) of cholecalciferol per day has been associated with signs of hypervitaminosis D in young children (AAP, 1963). Since the toxic level of vitamin D may in some cases be only 5 times the RDA, and there is evidence that sunlight-stimulated production of the vitamin is active throughout the warm months, dietary supplements may be detrimental for the normal child or adult who drinks at least two glasses of vitamin D-fortified milk per day (AAP, 1963).

References

AAP (American Academy of Pediatrics). 1963. The prophylactic requirement and the toxicity of vitamin D. Pediatrics 31:512–525.

AAP (American Academy of Pediatrics). 1985. Composition of Human Milk; Normative Data. Appendix K. Pp. 363–368 in Pediatric Nutrition Handbook, 2nd ed. American Academy of Pediatrics, Elk Grove Village, Ill.

Chesney, R.W., J. Zimmerman, A. Hamstra, H.F. DeLuca, and R.B. Mazess. 1981. Vitamin D metabolite concentrations in vitamin D deficiency. Are calcitriol levels normal? Am. J. Dis. Child. 135:1025–1028.

Chick, H., E.J. Dalyell, E.M. Hume, H.M.M. Mackay, H.H. Smith, and H. Wimberger. 1923. Studies of rickets in Vienna, 1919–1922. Medical Research Council Special Report Series, No. 77. Medical Research Council, London.

Clemens, T.L., S.L. Henderson, J.S. Adams, and M.F. Holick. 1982. Increased skin pigment reduces capacity of skin to synthesize vitamin D_3. Lancet 1:74–76.

DeLuca, H.F. 1979. Vitamin D. Metabolism and Function. Springer-Verlag, Berlin. 80 pp.

DeLuca, H.F. 1988. The vitamin D story: a collaborative effort of basic science and clinical medicine. FASEB J. 2:224–236.

Dent, C.E., and R. Smith. 1969. Nutritional osteomalacia. Quart. J. Med. 38:195–209.

Edidin, D.V., L.L. Levitsky, W. Schey, N. Dumbovic, and A. Campos. 1980. Resurgence of nutritional rickets associated with breast-feeding and special dietary practices. Pediatrics 65:232–235.

Egsmose, C., B. Lund, P. McNair, B. Lund, T. Storm, and O.H. Srensen. 1987. Low serum levels of 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D in institutionalized old people: influence of solar exposure and vitamin D supplementation. Age Ageing 16:35–40.

Finberg, L. 1981. Human milk feeding and vitamin D supplementation—1981. J. Pediatr. 99:228–229.

Fomon, S.J. 1974. Infant Nutrition, 2nd ed. W.B. Saunders, Philadelphia.

Fraser, D.R. 1988. Calcium-regulating hormones: vitamin D. Pp. 27–41 in B.E.C. Nordin, ed. Calcium in Human Biology. Springer-Verlag, London.

Glaser, K., A.H. Parmelee, and W.S. Hoffman. 1949. Comparative efficacy of vitamin D preparations in prophylactic treatment of premature infants. Am. J. Dis. Child. 77:1–14.

Greer, F.R., and R.C. Tsang. 1983. Vitamin D in human milk: is there enough? J. Pediatr. Gastroenterol. Nutr. 2:S227–S281.

Greer, F.R., J.E. Searcy, R.S. Levin, J.J. Steichen, P.S. Steichen-Asche, and R.C. Tsang. 1982. Bone mineral content and serum 25-hydroxyvitamin D concentrations in breast-fed infants with and without supplemental vitamin D: one year follow-up. J. Pediatr. 100:919–922.

Haussler, M.R., and T.A. McCain. 1977. Basic and clinical concepts related to vitamin D metabolism and action. N. Engl. J. Med. 297:1041–1050.

Hayward, L., M.T. Stein, and M.I. Gibson. 1987. Nutritional rickets in San Diego. Am. J. Dis. Child. 141:1060–1062.

Lawson, D.E.M. 1980. Metabolism of vitamin D. Pp. 93–126 in A.W. Norman, ed. Vitamin D: Molecular Biology and Clinical Nutrition. Marcel Dekker, New York.

Nordin, B.E.C. 1973. Metabolic Bone and Stone Disease. Williams and Wilkins Co., Baltimore. 309 pp.

Omdahl, J.L., P.J. Garry, L.A. Hunsaker, W.C. Hunt, and J.S. Goodwin. 1982. Nutritional status in a healthy population: vitamin D. Am. J. Clin. Nutr. 36:1225–1233.

Reeve, L.E., R.W. Chesney, and H.F. DeLuca. 1982. Vitamin D of human milk: identification of biologically active forms. Am. J. Clin. Nutr. 36:122–126.

Reid, I.R., D.J. Gallagher, and J. Bosworth. 1986. Prophylaxis against vitamin D deficiency in the elderly by regular sunlight exposure. Age Ageing 15:35–40.

Roberts, C.C., G.M. Chan, D. Folland, C. Rayburn, and R. Jackson. 1981. Adequate bone mineralization in breast-fed infants. J. Pediatr. 99:192–196.

Rosen, J.F., and R.W. Chesney. 1983. Circulating calcitriol concentrations in health and disease. J. Pediatrics 103:1–17.

Steichen, J.J., R.C. Tsang, F.R. Greer, M. Ho, and G. Hug. 1981. Elevated serum 1,25-dihydroxyvitamin D concentrations in rickets of very low-birth-weight infants. J. Pediatr. 99:293–298.

Stryd, R.P., T.J. Gilbertson, and M.N. Brunden. 1979. A seasonal variation study of 25-hydroxyvitamin D_3 serum levels in normal humans. J. Clin. Endocrinol. Metab. 48:771–775.

Tsang, R.C. 1983. The quandary of vitamin D in the newborn infant. Lancet 1:1370–1372.

USDA (U.S. Department of Agriculture). 1983. Table 2A-1.1. Milk, milk products; eggs; legumes, nuts, seeds. Average intake per individual per day, 1977–78. Pg. 126 in Nationwide Food Consumption Survey 1977–78. Food Intakes: Individuals in 48 States, Year 1977–78. Report No. I-1. Consumer Nutrition Division, Human Nutrition Information Service. U.S. Department of Agriculture, Hyattsville, Md.

USDA (U.S. Departament of Agriculture). 1986. Nationwide Food Consumption Survey Continuing Survey of Food Intakes by Individuals: Men 19–50 Years, 1 Day, 1985. Report No. 85-3. Nutrition Monitoring Division, Human Nutrition Information Service, U.S. Department of Agriculture, Hyattsville, Md. 94 pp.

USDA (U.S. Department of Agriculture). 1987. Nationwide Food Consumption Survey Continuing Survey of Food Intakes by Individuals: Women 19–50 Years and Their Children 1–5 Years, 4 Days, 1985. Report No. 85-4. Nutrition Monitoring Division, Human Nutrition Information Service. U.S. Department of Agriculture, Hyattsville, Md. 182 pp.

Webb, A.R., L. Kline, and M.F. Holick. 1988. Influence of season and latitude on the cutaneous synthesis of vitamin D_3: exposure to winter sunlight in Boston and Edmonton will not promote vitamin D_3 synthesis in human skin. J. Clin. Endocrinol. Metab. 67:373–378.